REMARKS

The amendments to Claims 1, 4, 6 and 7 are supported by original Claims.

The nucleotide sequence "CGAGGCCTGACGCGTGTACGTA" is described as base sequence of 1 to 22 of the sequence of SEQ ID NO: 1.

No new matter has been entered by the amendments.

Claims 1, 4, 6 and 7 are pending in this application.

The products that are trademarks are noted as such by including the ™ symbol. As indicated in MPEP 608.01(v) capitalization OR the ™ symbol is sufficient. Accordingly, no further amendments to the specification have been submitted.

The rejection of Claims 4 and 6-7 under 35 USC 112, second paragraph is no longer applicable as claim 4 has been amended to define the sequence that is in the StuI site, nucleotides 1-22 of SEQ ID NO: 1.

Withdrawal of the rejection is requested.

The rejection of Claims 1-7 under 35 USC 112, first paragraph ("written description") is believed to be no longer applicable to the claims as amended here. The claims have been amended a to be a CMV-Y vector having RNA2 encoding a 2b protein of RNA2 deleted from StuI to the stop codon of an ORF (2bORF) encoding the 2b protein. Such is described on pages 6-9 and in the Examples beginning on page 11 of the specification. Therefore, possession of the subject matter claimed is found in the specification as filed.

In a similar manner the rejection under 35 USC 112, first paragraph relating to enablement is believed to be no longer applicable as the claims re defined to be not any virus

vector but a CMV-Y vector having a specified deletion. As discussed above, such is described in the specification and therefore would be enabled by that description as well.

Withdrawal of the rejections is requested.

The rejection of Claim 1 under 35 USC 102(b) citing Ding is no longer applicable as Claim 1 has been amended to define the vector as a CMV-Y vector containing RNA2 deleted from StuI to the stop codon of 2bORF. In contrast, Ding et al. describes a CMV in which the 2b protein gene of RNA2 has been substituted with 2b protein of tomato aspermy virus, that is the entirety of the 2bORF has been deleted and substituted (see Abstract, the paragraph bridging col. 1-2 of page 7470, and Fig. 1).

Withdrawal of the rejection is requested.

The rejection combining Ding and Soards is not sustainable because the art does not teach that which is claimed.

It is acknowledged in the rejection that Ding does not describe a CMV-Y wherein a region from a StuI site to a stop codon of an ORF encoding 2b protein of RNA2. Soards allegedly makes up for that deficiency but in fact it does not.

Ding's constructs are CMV substituted with the 2bORF of TAV and are reported as having considerable potent toxicity that causes infected plants to wither (CMV-qt of Table 2 resulted in +++ = severe systemic symptoms in all plants). This prevents Ding's construct from being useful as a vector.

Soards et al. is a CMV-Fny strain (Fny-CMVΔ2b) in which a sequence encoding 2b protein encoded in 2419-2713 of CMV RNA2, which is the entire nucleotide sequence encoding 2b protein, has been deleted. As is stated in lines 13 to 15 of col. 1 on page 648 of

Soards: "Fny-CMVΔ2b only exhibits accumulation of a low level of CP (coat protein) at all times", and in the heading in lines 3 to 5 of the col. 2 on that same page: "Fny-CMVΔ2b exhibits slower progression than the wild strain", the efficiency of both infectivity and gene expression following transformation decreases. Soards et al. also states on page 649 that CMVΔ2b-GFP only exhibits limited spreading (line 11 of the left column and lines 8 and 9 of the Discussion of col. 2), and that deletion of 2bORF can effect virus replication (lines 17 and 18 of the Discussion of the col. 2).

Therefore when taken together Ding teaches that symptoms may be enhanced when 2b protein is substituted between different CMV and Soards et al. merely indicates that virus infectivity and propagation decrease when 2b protein is deleted. Neither, however, teaches how to prepare plant virus vector using CMV-Y that is useful for plant transformation.

In contrast, the CMV-Y vector defined in the claims is a plant virus vector that demonstrates the remarkable and particularly preferable effect of not losing virus systemic infectivity, results in slower progression of symptoms than wild-type CMV-Y and allows an exogenous gene to be stably expressed.

Applicants submit herewith a publication of Otagaki et al. (Plant Biotechnology, 2006, Vol. 23, pp. 259-265) as a reference indicating these advantages of the CMV-Y vector as claimed.

As described in the Otagaki publication, the CMV-Y vector (CMV2-A1 in Figure 1) only exhibits mild symptoms even when infected into *N. benthamiana* (lines 31 to 33 of the col. 2 on page 254), and has the characteristic of spreading throughout the entire plant, and this is also described in the specification (e.g., see page 8, 1st paragraph, page 9, 2nd paragraph, and the "Results" of the Example on page 28 in the specification).

There is nothing provided by the combination of Ding and Soards that CMV-Y, in which the 2bORF of RNA2 from StuI to the stop codon of the 2bORF has been partially

deleted, demonstrates ideal properties as a vector. This must be so as Ding describes a CMV in which toxicity is merely enhanced by substituting the entire 2bORF of CMV with 2bORF of TAV, and Soards who describes that CMV in which virus infectivity and spread decreased as a result of completely deleting the 2bORF of CMV-Fny.

Withdrawal of the rejection is requested.

The rejection combining Ding and Soards with Roossinck is also not sustainable even when considered with Roossink et al. indicating that CMV-Y and CMV-Fny are related species. That is, there is nothing in this combination of art that CMV-Y, in which the 2bORF of RNA2 from StuI to the stop codon of the 2bORF has been partially deleted, demonstrates ideal properties as a vector. This must be so as Ding describes a CMV in which toxicity is merely enhanced by substituting the entire 2bORF of CMV with 2bORF of TAV, and Soards who describes that CMV in which virus infectivity and spread decreased as a result of completely deleting the 2bORF of CMV-Fny.

Withdrawal of the rejection is requested.

The rejection combining Ding and Soards with Ding (1995) is also not sustainable even when considered with Ding teaches merely indicates that a stop codon was created within 2bORF without altering the amino acids of the 2a protein of RNA2. That is, there is nothing in this combination of art that CMV-Y, in which the 2bORF of RNA2 from StuI to the stop codon of the 2bORF has been partially deleted, demonstrates ideal properties as a vector. This must be so as Ding describes a CMV in which toxicity is merely enhanced by substituting the entire 2bORF of CMV with 2bORF of TAV, and Ding (1995) who describes that CMV in which virus infectivity and spread decreased as a result of completely deleting the 2bORF of CMV-Fny.

Withdrawal of the rejection is requested.

A Notice of Allowance is requested.

Respectfully submitted,

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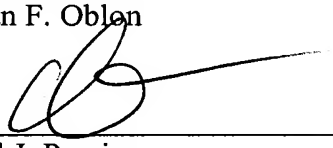
Norman F. Oblon



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